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*NICOTINIC ACID – KEY VITAMIN IN PELLAGRA**

The years 1915 to 1937 marked the successful culmination of a quest begun many decades before and having as its goal the answer to the cause and cure of pellagra. In this relatively brief twenty-two year period, four signal accomplishments were made: (1) The disease was experimentally produced and cured by dietary means alone, thereby establishing it beyond question as a disease of nutritional origin. (2) The separation of the heat-labile anti-beriberi fraction (vitamin B_1) from "vitamin B" indicated the multiple nature of "vitamin B" and pointed to the existence of a fraction other than vitamin B_1 as the active anti-pellagra factor. (3) Goldberger's observation that canine blacktongue is the counterpart of human pellagra provided an experimental animal and method of study which materially stimulated progress in the search for the active factor. (4) The discovery by Elvehjem and his collaborators of nicotinic acid as the primary protective and curative factor in the treatment of canine blacktongue suggested the use of this compound in the treatment of human pellagra and led to the final solution of the pellagra problem.

Clinical Studies with Nicotinic Acid

Immediately following the work of Elvehjem, Madden, Strong and Woolley (1), many research workers investigated the use of nicotinic acid and nicotinic acid amide as therapeutic agents in pellagra. A few months after the publication of the work of Elvehjem *et al,* the first published report appeared describing the successful treatment of four cases of human pellagra with nicotinic acid (2). This was followed by a report by Smith and collaborators (3) of a man with a fifteen year history of recurrent pellagra, presenting severe symptoms including dementia, who exhibited dramatic improvement within twenty-four hours following treatment with nicotinic acid. Continued administration of nicotinic acid in 60 milligram doses per day resulted in a complete cure after twelve days. Spies *et al* (4, 5) reported rapid cure of the characteristic dermatitis, and of the painful tongue and mouth inflammation in a large number of pellagra patients, using either nicotinic acid, nicotinic acid amide or sodium nicotinate. They also

*The first section of this paper appeared in the October issue of *Borden's Review of Nutrition Research.*

reported dramatic restoration of mental faculties, improved appetite and return of normal intestinal function. These workers found, however, that the peripheral neuritis commonly associated with pellagra failed to respond to nicotinic acid therapy, requiring instead, specific supplementation with vitamin B_1. This finding constituted one of the earliest indications as to the multiple deficiency nature of pellagra. The following year and a half period witnessed more than a dozen similar reports describing the same dramatic responses obtained with nicotinic acid or its amide in the treatment of hundreds of cases of pellagra. These reports clearly established that all of the so-called classic symptoms of pellagra, including the diagnostic triad popularly referred to as the "three D's" – dermatitis, diarrhea and dementia — respond specifically to treatment with nicotinic acid.

Symptomatic Responses to Nicotinic Acid Therapy: The first noticeable effect of the administration of nicotinic acid in pellagra is the relief of glossitis (inflammation of the tongue) . The characteristic fiery redness and swelling usually disappear within a day. Ulcers which often appear below the tongue and on the lips, heal in less than a week, with striking relief from soreness and pain in the mouth. The inflammation affecting the oral mucous membranes frequently extends throughout the entire alimentary canal resulting in severe pain, nausea, abdominal distress, diarrhea and related symptoms. Most of these respond to treatment with nicotinic acid preparations after a few days. Loss of appetite is usually a symptom of thiamin deficiency as well and necessitates supplementation of nicotinic acid therapy with vitamin B_1.

The skin lesions associated with pellagra have been classified into six distinct types (6) . It was found (7, 8) that certain of these lesions, namely those which appear on the exposed surfaces of the body, are precipitated and aggravated by exposure to sunlight. The other types of lesions may appear on the same areas, or on other parts of the body, but are not influenced by exposure to the sun. Most of these lesions respond to nicotinic acid therapy within twenty-four hours; some may require several days. Sebrell (9) and Sydenstricker (10) and associates have reported the presence of additional lesions, particularly about the eyes and mouth, which do not respond to treatment with nicotinic acid and which they have been able to cure by administration of riboflavin. Here again the multiple deficiency nature of pellagra is emphasized.

The disappearance of the acute mental symptoms of pellagra has been described as the most dramatic response of pellagrins to nicotinic acid therapy and has been reported by numerous clinical investigators. Spies *et al* (11) state:

"These symptoms, varying from slight confusion to delirium and mania, disappear rapidly, often over night. The maniacal patients become calm and the confused patients, mentally clear. After therapy they become readjusted, and often have excellent insight and memory of their actions, ideas, and surroundings during the psychotic period. Apathy and lassitude give way to interest. In sharp contrast to the prompt and beneficial response of the mental symptoms to nicotinic acid is the lack of

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improvement in symptoms arising from the peripheral nervous system. The administration of crystalline vitamin B_1, however, gives prompt relief from these symptoms."

Associated Vitamin Deficiencies in Pellagra:

Some difference in opinion has existed as to whether pellagra is a multiple vitamin deficiency disease or whether it may be considered as a specific nicotinic acid deficiency. As expressed by Smith (6):

"A pure vitamin deficiency in man is exceedingly rare. The patient with rickets often has a vitamin A deficiency as well as a vitamin D deficiency, and the patient with scurvy may have beriberi as a complication, as was the case in the old disease known as 'ship beriberi.' Pellagrins frequently have associated deficiencies of vitamins B_1, A, C, B_6, riboflavin, proteins, and mineral salts. The cardinal symptoms of pellagra are cured by nicotinic acid, and we feel very strongly that pellagra should be regarded as a nicotinic acid deficiency. The associated deficiencies should be recognized when they appear and treated accordingly."

Clinical experience has shown, however, that the overwhelming majority of pellagrins subsisting on their customary limited diets invariably present evidence of secondary deficiencies associated especially with lack of thiamin and riboflavin. As has already been pointed out, the peripheral neuritis commonly occurring with pellagra is actually a symptom of thiamin insufficiency and, as such, will not respond to treatment with nicotinic acid. Loss of appetite (anorexia) associated with pellagra may be treated successfully with nicotinic acid, but unless vitamin B_1 is also administered, anorexia may recur after the initial stimulation of appetite. The need for riboflavin, as well, has been indicated by a number of investigators who have shown that this vitamin is the only successful means of treating many of the skin and ocular disturbances in pellagra. According to Elvehjem (14):

"Nicotinic acid will cure or prevent all conditions produced by a true nicotinic acid deficiency but can be of no value in the treatment of conditions produced by a deficiency of other factors and its value in the treatment of pellagrins is much more effective when all other factors are supplied in optimal amounts."

Spies *et al* (11) have enumerated the specific symptoms of pellagra which will respond to nicotinic acid therapy, stating as follows:

"Nicotinic acid or one of its closely related compounds, when administered in adequate amounts, has a prompt and beneficial effect on certain symptoms of clinical and subclinical pellagra. In cases of acute or chronic pellagra in relapse it will: (a) cause fading of the fiery red lesions of the mucous membranes and diminish the Vincent's infection associated with it, (b) in most cases, restore to normal disturbed gastrointestinal function, (c) restore to normal the mental function deranged moderately or severely in acute pellagra, (d) cause fading of the dermal erythema but not cure chronic changes of the skin. In cases of subclinical pellagra, the vague ill defined symptoms disappear and in persons subject to recurrences of the disease the development of clinical pellagra in prevented. In both clinical and subclinical pellagra, the sense of well-being, one of the attributes of health, is restored.

"Nicotinic acid, thiamin, and riboflavin are invaluable in treating the pellagrin who has a deficiency of thiamin and flavin. Detailed studies of the food consumed by pellagrins reveal inadequacies of most of the essential nutrients. It is imperative, therefore, to stress the fact that these chemical substances, although invaluable as therapeutic agents, cannot be expected to replace a liberal and well-balanced diet."

From the foregoing, it is apparent that nicotinic acid is indeed the key vitamin in the treatment of pellagra and that the so-called cardinal symptoms of the disease are directly attributable to a deficiency of this vitamin.

Nevertheless, it is important to keep in mind the fact that pellagrins very frequently develop associated deficiencies due to insufficiency of thiamin and riboflavin, so that effective therapy in pellagra must include all three of these B vitamins, and probably other nutritional factors as well. Typical deficiencies other than nicotinic acid reported in common dietaries of pellagrins are given in the following table.

A Comparison of the Nutritive Values of the Dietaries of Pellagrins with Standards for Normal People (11)

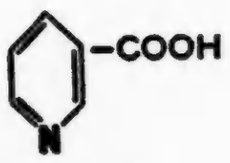
Nutrient	Calories	Protein	Minerals			Vitamins			
			Ca (gm.)	P (gm.)	Fe (mg.)	A (I.U.)	B (I.U.)	C (I.U.)	G (Sherman-Bourquin units)
Standard* 19 men	3000	67	.68	1.32	15	5600	150–385	150–375	600–800
27 women	2500	75	.88	1.20	13–15	5600	125–300	125–300	500–800
3 children									
boy—13–15 yrs.	3000	75	.88	1.32	15	5600	150–385	150–375	600–800
boy—4–6 yrs.	1500	55	1.00	1.00	8–11	4200	75–188	100–250	300–650
child under 4 yrs.	1200	45	1.00	1.00	6–9	4200	60–150	100–250	240–600
Per cent patients below standard	97	95	83	85	98	93	95	42	94
Range in per cent below standard	17–91	5–86	7–92	5–92	7–90	1–100	20–100	4–100	17–100
Average per cent below standard	35	50	61	58	51	67	72	47	73

* From "Quantities of Nutrients for Individuals per Day to Be Used in Evaluating the Adequacy of a Diet"—Dr. Hazel Stiebeling, Bureau of Home Economics, U. S. Dept. Agriculture.

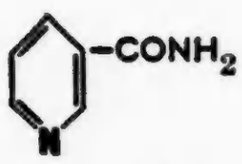
For this reason, the importance of supplying foods rich in thiamin and riboflavin, as well as in nicotinic acid, cannot be overemphasized in the prevention and cure of pellagra. Thus, the rationale of the early pellagra-preventive diet proposed by Goldberger — meat, milk, eggs, fruits and vegetables — becomes apparent in the light of present-day knowledge concerning the prevention of deficiencies involved in the development of pellagra.

Pyridine Derivatives in the Treatment of Pellagra: In a continuation of their studies on the pyridine derivatives, nicotinic acid and nicotinic acid amide, Elvehjem and collaborators (12) tested a number of other pyridine derivatives for their antiblacktongue potency.

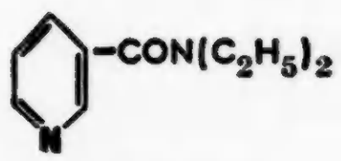
Pyridine



Nicotinic Acid
(Niacin)



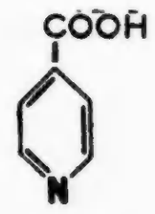
Nicotinic Acid Amide
(Nicotinamide; Niacin Amide)



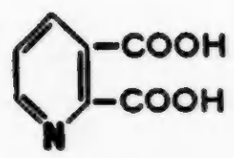
Nicotinic Acid Diethylamide
(Coramine)

Fig. I Pyridine Compounds Possessing Antiblacktongue Activity

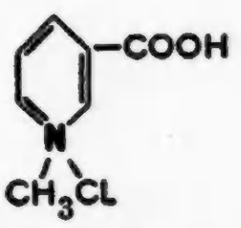
They found (1) that the nicotinic acid or nicotinic acid amide structure is essential for antiblacktongue potency and (2) that only those compounds which are capable of conversion to these substances in the body (by oxidation or hydrolysis) possess similar potency (see Fig. 1). On the other hand, even such closely related compounds as isonicotinic acid exhibit no antiblacktongue activity because physiological conversion to the nicotinic acid (or amide) form is not possible (See Fig. 11).



Isonicotinic Acid



Quinolinic Acid



Trigonelline Hydrochloride

Fig. II Pyridine Compounds Possessing No Antiblacktongue Activity

A number of the active compounds have been tried on patients with pellagra (13). In many instances, results similar to those obtained with the dog were obtained. Sufficient experimental data are not yet available, however, to justify the clinical use of any of the derivatives of nicotinic acid with the exception of nicotinic acid amide and the diethyl amide of nicotinic acid (known as coramine). In this connection, it is interesting to note that nicotinic acid amide and coramine have been found to have certain clinical advantages in that their administration does not result in the flushing and tingling sensations which sometimes follow the administration of nicotinic acid.

Physiology and Metabolism of Nicotinic Acid:

Physiological Significance: Nicotinic acid, in the form of its amide, is now known to be an integral part of two enzyme systems within the body, namely:

coenzyme I (diphosphopyridine nucleotide) and coenzyme II (triphosphopyridine nucleotide). These coenzymes are intimately involved in the vital mechanisms of intercellular oxidation, being especially concerned with the metabolism of proteins and carbohydrates. It has been demonstrated (15, 16) that the coenzyme level is actually reduced in the tissues of dogs with experimental blacktongue. It is believed that a marked reduction in the coenzyme content of blood, brain or adrenal cortex may result in death. Experimental evidence indicates that nicotinic acid probably has important physiological functions in the living animal in addition to that of furnishing building material for certain coenzymes. Some of these possible additional functions of nicotinic acid are thought to be indirect regulation of water metabolism, and of the metabolism of the heavy metals; regulation of iron-porphyrin metabolism with consequent influence upon certain types of anemia; and influence upon motility of the stomach and small intestine (16a). These postulated functions await experimental confirmation. The precise mechanisms involved in the production of the various symptoms and disorders characteristic of pellagra due to insufficiency of nicotinic acid are also not yet clearly established.

Nomenclature: As described in Part I of this REVIEW (October, 1943) nicotinic acid may be derived from nicotine, an ingredient of tobacco leaves, by oxidation. The process involves the use of powerful oxidizing agents which break up the highly complex nicotine molecule, yielding a new material with entirely different properties, namely the vitamin, nicotinic acid. Although nicotinic acid was originally produced by this method, the vitamin is now synthesized by many other methods, one of the more common being the synthesis from pyridine. Chemically, nicotinic acid, is called *pyridine-beta-monocarboxylic acid* (see formula, page 5).

Because of the frequent erroneous association in the popular mind of the vitamin, nicotinic acid, with the alkaloid, nicotine of tobacco, the Food and Nutrition Board of the National Research Council recommended the adoption of the name, *niacin,* as a synonym for this vitamin. In January, 1942 the Federal Security Agency officially approved this recommendation, adopting the terms *niacin* and *niacin amide* as common names for food and drug label declaration purposes, for the vitamin substances scientifically designated respectively as nicotinic acid and nicotinic acid amide.

Normal Dietary Requirements for Niacin: The following table sets forth the recommended daily allowances for nicotinic acid (niacin) for various age groups of both sexes as promulgated by the Food and Nutrition Board of the National Research Council:

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RECOMMENDED DIETARY ALLOWANCES
FOOD AND NUTRITION BOARD, NATIONAL RESEARCH COUNCIL
January 1943

	Niacin (Nicotinic Acid) mg.
Man (70 Kg.)	
Sedentary	15
Moderately active	18
Very active	23
Woman (56 Kg.)	
Sedentary	12
Moderately active	15
Very active	18
Pregnancy (latter half)	18
Lactation	23
Children up to 12 years:	
Under 1 year†	4
1–3 years‡	6
4–6 years	8
7–9 years	10
10–12 years	12
Children over 12 years:	
Girls, 13–15 years	14
16–20 years	12
Boys, 13–15 years	16
16–20 years	20

Food Sources of Niacin:

Closely following the recognition of the biological importance of nicotinic acid, numerous analytical methods were devised for determining, quantitatively, the presence of this vitamin in food. The early work of Goldberger, and of Elvehjem employed the dog as an experimental biological tool, by means of which foods were assayed for nicotinic acid potency in terms of their relative ability to cure blacktongue. The biological method has now been supplemented by chemical and microbiological methods which offer increased speed and accuracy in the quantitative estimation of nicotinic acid. The history and description of these methods constitute in themselves a most interesting subject, but they are beyond the scope of this review.

The perfection of these methods has made available an extensive amount of data on the niacin content of many common foods. To date, a number of reports have appeared in the literature giving valuable, detailed information on the niacin content of milk and milk products (17), meat and meat products (18, 19, 20), cereal products (21), fruits and vegetables (22) and other common foodstuffs (23, 24, 25). In general, meat, poultry, peanuts and yeast are the most important common sources of the vitamin; cereals contribute significantly; fruits and vegetables are rather poor sources.

Milk is an important anti-pellagra food, although its relative niacin content is not very high. The value of milk in the anti-pellagra diet lies in the fact that the niacin content of milk is only one of its many important nutritional factors. Since the diet of pellagrins has been demonstrated to be deficient not only in the B vitamins, but in vitamin A, protein, calcium and other minerals, and, to a lesser extent, vitamin C (N.B. see table on page 4), it is apparent that milk, which supplies generous amounts of many of these essentials, necessarily takes an important place in the anti-pellagra diet.

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†Needs of infants increase from month to month. The amounts given are for approximately 6–8 months.
‡Allowances are based on needs for the middle year in each group (as 2, 5, 8, etc.) and for moderate activity.

FOODS SUPPLYING A FULL DAY'S QUOTA OF PELLAGRA-PREVENTIVE VALUE (26)

A quart of milk or buttermilk;
A pint of evaporated milk;
One-third to one-half pound
of dried skimmed milk,
of lean meat,
of canned salmon,
of peanut meal, or
of wheat germ;

One pound of fresh or canned collards,
of kale,
of green peas, or
of turnip greens; or
Two to three pounds of tomatoes, fresh
or canned, or
of tomato juice

Conclusion:

Though many decades were spent in vain attempts to solve the mystery of pellagra — one of the greatest of human scourges — scientists have at last succeeded in finding the key to the origin and elimination of this disease. Pellagra has been established as a nutritional deficiency disease and nicotinic acid (niacin) has been shown to be the key vitamin involved in its prevention and cure. The importance has been stressed of other members of the vitamin B complex, particularly thiamin and riboflavin, as essential supplementary factors in the prevention and treatment of pellagra. In terms of practical daily nutrition, this means that the elimination of pellagra is dependent on the ample consumption of such foods as milk, cheese, meat, eggs, fish, peanut and soybean products which provide not only niacin, but riboflavin and thiamin as well.

The government food fortification program, which requires the addition of niacin, thiamin and riboflavin to white flour and other dietary staples, is but an initial step toward achieving the goal of adequate nutrition for all of the populace. The selection and consumption of the right kinds and amounts of foods by all individuals are the final goals which must be attained before the scientific conquest of pellagra, and other nutritional deficiency diseases, can become a completed conquest in terms of the eradication of these diseases.

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NEWS DIGEST

AN ANTI-THIAMIN FACTOR now takes its place, along with avidin, the anti-biotin factor, as another anti-vitamin substance recently uncovered by research scientists. The anti-thiamin factor has not yet been completely identified chemically. It is a protein-like material which is found in raw fish and is capable of destroying thiamin apparently by enzymatic action.

A thiamin deficiency disease, known as Chastek paralysis which develops in foxes eating a feed mixture apparently adequate in thiamin and containing raw fish as an ingredient, led to the discovery of the anti-thiamin factor. Chastek paralysis is believed to be the counterpart of Wernicke's syndrome — a human disease characterized by inflammation and hemorrhage of the brain. Recent research has shown that the anti-thiamin factor is destroyed by cooking and by peptic digestion, so that fish meal eaten alone, either in the *raw* or *cooked* state, does not result in thiamin destruction. Raw fish-meal, however, when in contact with other foods before ingestion, as in an animal feed mixture, inactivates any thiamin present in the foods.

Because the anti-thiamin factor is destroyed in cooking, drying or digestion, the only opportunity for its significantly affecting thiamin nutrition in man occurs when the major thiamin-containing constituents of the diet are mixed with raw whole fish and allowed to stand at room temperature for some time before being cooked, dried or eaten. Under ordinary circumstances such preparation of human foods is rare. Further studies may yield valuable information as to possible similar anti-vitamin mechanisms in man.

—1101

BETAINE, a completely methylated aminoacetic acid, has been known as an effective donor of methyl groups since the recent experimental work leading to the discovery of transmethylation as a vital process in human and animal metabolism. For some time it has been thought that betaine was effective only in man and other mammals. It has now been demonstrated, however, to be capable under certain conditions of exerting a growth effect in the chick equivalent to that of choline. The new data indicate that betaine in certain respects may assume the physiological functions of choline or methionine in the chick. Betaine or a derivative may be a physiological requirement which can be formed, if necessary, from choline or methionine. Betaine occurs in appreciable quantities in beet sugar molasses.

—1102

DEFATTED CORN GERM may soon be commercially developed as a new source of protein for human consumption to supplement present dwindling supplies of available meat protein. A preliminary report indicates that a new kind of palatable "defatted" corn germ having good keeping qualities, has been tested on laboratory animals with highly successful results. It is stated that except for the cashew nut, defatted corn germ is the only plant food thus far studied by the investigators which supplies protein of as high a biological value as lean meat.

According to tests, the defatted corn germ contains 21 percent protein which is 85 percent as digestible as beef protein and equally as high as beef protein in biological value. It is said to be rich in vitamin and mineral content, having an unusually high iron value of 300 parts per million. Estimations are that sufficient defatted corn germ may be made available to yield an annual supply of one-half billion pounds of valuable protein for human use.

—1103

VITAMIN B_c, the so-called chick anti-anemia vitamin, discovered a few years ago, is now being investigated to determine its chemical properties. Lack of the vitamin induces a special type of severe anemia in young chicks which develops usually in the first three to five weeks of life. A water extract of liver has been found to be an excellent source of the new vitamin.

—1104

WHOLE SUGAR CANE AND SUGAR CANE JUICE have been found to be sources of the B vitamins. Inasmuch as sugar constitutes one of the cheapest sources of energy foods in the United States, a study of the vitamin content of whole sugar cane and sugar cane juice as produced in Louisiana and Cuba was undertaken. Based on each pound of sucrose in the whole mature Cuban cane stalk and in the sugar cane juice, the following vitamin values are reported:

	Whole Cane		Juice	
	Aver.	Max.	Aver.	Max.
	mg.	mg.	mg.	mg.
Thiamin	2.05	17.95	0.50	1.1
Riboflavin	1.1	5.7	0.215	0.4
Pantothenic acid	6.0	38.6	4.4	9.9
Niacin	5.3	52.25	2.0	3.3
Biotin	0.2	0.7	0.08	0.1

The corresponding values for Louisiana cane were lower for thiamin, riboflavin and biotin, higher for pantothenic acid, and of the same order for niacin. In general, it was concluded that whole mature cane is a fair source of thiamin and riboflavin, a rich source of pantothenic acid and a good source of niacin.

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